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Comparison of Effects of Moderate Doses of Rosuvastatin and Atorvastatin on Plaque Regression in Patients with Non-intervened Intermediate Coronary Stenosis

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Background: Lipid-lowering therapy with statins improved clinical outcomes, survival and reduced the progression of atherosclerosis.
Objectives: We used serial intravascular ultrasound (IVUS) to compare the effects of moderate doses of rosuvastatin and atorvastatin on plaque regression in Korean patients with intermediate coronary stenosis.
Methods: This study was a prospective, randomized, and comparative study for lipid lowering therapy with rosuvastatin 20 mg (n=65) and atorvastatin 40 mg (n=63) using serial IVUS examinations (baseline and 11-month follow-up). Efficacy parameters included changes in total atheroma volume (TAV) and percent atheroma volume (PAV) from baseline to follow-up. TAV was calculated by summation of atheroma area from each measured image as: TAV=∑ (EEM area-lumen area). The PAV was determined using the formula: PAV=100 X [∑ (EEM area-lumen area) / ∑ (EEM area)]. Plaque progression or regression was determined by volumetric IVUS analysis at the target lesion.
Results: Follow-up low-density lipoprotein cholesterol (LDL-C) (62±20 vs. 70±24 mg/dl, p=0.053) and high-sensitivity C-reactive protein (hs-CRP) (0.10±0.14 vs. 0.10±0.18 mg/dl, p=1.000) were not significantly different and changes of LDL-C (-60±33 vs. -47±39 mg/dl, p=0.057) and hs-CRP (-0.40±1.18 vs. -0.84±2.07 mg/dl, p=0.16) from baseline to follow-up were not significantly different between rosuvastatin and atorvastatin groups. Changes of TAV (-4.4±7.3 vs. -3.6±6.8 mm^3, p=0.5) and PAV (-0.73±2.05 vs. -0.19±2.00 %, p=0.14) from baseline to follow-up were not significantly different between rosuvastatin and atorvastatin groups. Plaque was regressed in 85% in rosuvastatin group and in 70% in atorvastatin group at follow-up (p=0.064). Plaque progressors had higher baseline hs-CRP (1.28±2.70 vs. 0.54±1.16 mg/dl, p=0.034) and higher follow-up LDL-C (78±24 vs. 63±21 mg/dl, p=0.002) compared with plaque regressors. Follow-up LDL-C (OR 1.038, 95% CI 1.003-1.060, p=0.036) and baseline hs-CRP (OR 1.025, 95% CI 1.001-1.059, p=0.046), not the type of statin, were the independent predictors of plaque progression at follow-up.
Conclusions: Moderate doses of rosuvastatin and atorvastatin could contribute to the effective plaque regression and follow-up LDL-C and baseline hs-CRP are associated with plaque progression in Korean patients with intermediate coronary stenosis.

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Long-term Impact of Concomitant Use of Proton Pump Inhibitors (PPIs) and Clopidogrel Following Percutaneous Coronary Intervention

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Background: Dual antiplatelet therapy, consisting of aspirin and clopidogrel, has been the cornerstone of pharmacotherapy following percutaneous coronary intervention (PCI). PPIs have been concomitantly used with aspirin and clopidogrel to reduce the risk of gastrointestinal bleeding complications. However, recent pharmacodynamic studies demonstrated that PPIs might attenuate clopidogrel-mediated platelet inhibition, through their effects on the hepatic cytochrome P450 CYP2C19 system. We investigated the clinical significance of concomitant PPI and clopidogrel use on long-term all-cause mortality in a population of patients undergoing PCI.
Methods: In a retrospective observational study, using the 2004/2005 Cornell Angioplasty Registry Database, we evaluated short- and long-term adverse events in 2,504 patients, undergoing elective or urgent PCI. Patients were divided into 2 groups: 1) those discharged on a PPI; or 2) discharged without PPI therapy after PCI. Patients presenting with an acute ST-elevation myocardial infarction (MI) ≤24 hours, thrombolytic therapy ≤7 days, hemodynamic instability/ shock, or renal insufficiency were excluded. The mean follow-up period was 4.5 years.
Results: Of the study cohort, 726 patients (31%) were discharged on a PPI plus clopidogrel and 1,650 patients (69%) were discharged on clopidogrel without PPIs. The incidence of in-hospital mortality (0.1% vs. 0.2%, p=0.731), emergent PCI (0.4% vs. 0.1%, p=0.088), emergent CABG (0.1% vs. 0%, p=0.306), stroke (0.3% vs. 0.1%, p=0.223), and major adverse cardiovascular events (5.4% vs. 7.3%, p=0.091) was similar in the PPI vs. no PPI group, respectively. There was a lower incidence of post-procedural myocardial infarction (5.1% vs. 7.1%, p=0.007) in the PPI plus clopidogrel group. At follow up, all-cause mortality was similar in the PPI vs. no PPI group (12.7% vs.10.9%, HR 1.19, 95% CI 0.92-1.53, p=0.184). After a propensity score-adjusted multivariate Cox regression analysis, PPI use was not associated with an increased risk of long-term mortality (hazard ratio 1.04, 95% CI 0.74-1.48, p=0.814).
Conclusions: Concomitant administration of clopidogrel and PPIs is not associated with an increased risk of long-term all-cause mortality following PCI.

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The Impact of Chronic Statin Therapy on Peri-procedural Myocardial Infarction in Patients Undergoing Percutaneous Coronary Intervention

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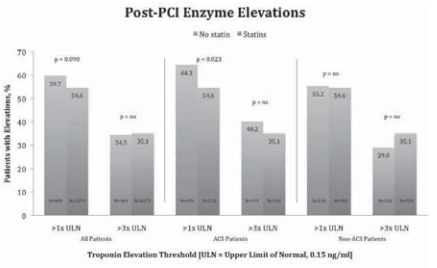
Background: Peri-procedural statin therapy has been shown to reduce the rate of post-PCI myocardial infarctions. However, the impact of chronic statin therapy on post-procedural myocardial infarction remains unknown. We examined the impact of long-term statin therapy on post-PCI cardiac enzyme elevations (cTnI and CK-MB) in patients undergoing non-emergent PCI.
Methods: Using the 2004/2005 Cornell Angioplasty Registry, we examined 1,482 patients undergoing non-emergent PCIs, who had normal pre-procedural cTnI or CK-MB levels, and whose charts contained information regarding pre-admission statin therapy. The population was divided into two groups: 1) patients on chronic (≥7 days) statin therapy prior to PCI (n=1,073); and 2) patients not on chronic statin regimen (n=409). Post-PCI cardiac enzymes were assessed before and 8, 16 and 24 hours after PCI (CK-MB upper limit of normal [ULN] - 4.5 ng/ml and cardiac troponin I ULN - 0.15 ng/ml). Results: There was a trend towards a lower incidence of cTnI elevations ≥1x ULN in patients on chronic statin therapy versus those not receiving pre-procedural statins (54.6% vs. 59.7%, respectively, p=0.090). There was no difference in the incidence of cTnI elevations ≥3x ULN in patients on chronic statin therapy versus those not on statins at home in all patients (35.1% vs. 34.5%, respectively, p=0.855) and in a subgroup of patients with ACS (35.1% vs. 40.2%, respectively, p=0.226) (Figure). The incidence of CK-MB elevations ≥1x ULN or ≥3x ULN, as well as peak cTnI and CK-MB levels, were similar between two groups.



Conclusions: Chronic statin therapy does not reduce the incidence of peri-procedural myocardial infarction in patients undergoing non-emergent PCI. These findings support a strategy of routine reloading with high-dose statins early before PCI on the background of chronic statin therapy.

Peripheral Vascular Intervention

(Abstract Nos 399-418)

TCT-399

Long-term Results Of Endovascular Aneurysm Repair (EVAR) Versus Open Repair In Patients With Large Abdominal Aortic Aneurysm: Results Of The UK EVAR Trial 1

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Long-term results of EndoVascular Aneurysm Repair (EVAR) versus open repair in patients with large abdominal aortic aneurysm: results of the UK EVAR Trial 1
Background: Limited data exist on the long-term performance of endovascular aneurysm repair (EVAR) compared with open repair of abdominal aortic aneurysm.
Methods: Between September 1999 and 2004, 1252 patients with large aneurysm (≥5.5cm) were randomised to receive either EVAR (n=626) or open repair (n=626) across 37 UK hospitals. Patients were followed for mortality, complications, re-interventions and costs until December 2009 (min follow-up 5 years). Logistic and Cox regression modelling were used to compare these outcomes between randomised groups.
Results: A significant reduction in 30-day operative mortality was seen in the EVAR group relative to the open repair group; 1.8% versus 4.3% respectively; adjusted odds ratio 0.39 [95% CI 0.18-0.87], p=0.021. This early benefit in aneurysm-related mortality was maintained for a number of years but was eroded by the end of the study; adjusted hazard ratio 0.92 [95% CI 0.57-1.49], p=0.731, due in part to fatal endograft ruptures. By the end of follow-up, no significant difference was seen between the groups in terms of all-cause mortality; adjusted hazard ratio 1.03 [95% CI 0.86-0.721], p=0.721. The rates of graft-related complications and re-interventions were higher in the EVAR group and the incidence of new complications continued out to 8 years, contributing to higher overall costs.
Conclusions: EVAR was associated with a significantly lower operative mortality but late endograft ruptures appeared to erode this early survival benefit such that no differences are seen in all-cause or aneurysm-related mortality in the long-term. Since EVAR was associated with increased complications and re-interventions it was a more expensive treatment option.

TCT-400

Long-term Results Of Endovascular Aneurysm Repair (EVAR) In Patients Considered Unfit For Open Repair: Results Of The UK EVAR Trial 2

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Background: Endovascular aneurysm repair (EVAR) was developed for patients with abdominal aortic aneurysm who were considered as unfit for open repair. EVAR Trial 2 is the only randomised trial investigating whether EVAR improves mortality in these patients.
Methods: Between September 1999 and 2004, 404 patients with large aneurysm (≥5.5cm) considered unfit for open repair were randomised to receive either EVAR (n=197) or no intervention (n=207) across 33 UK hospitals. Patients were followed for mortality, complications, re-interventions and costs until December 2009 (minimum follow-up 5 years). Cox regression modelling was used to compare outcomes between randomised groups.
Results: 30-day operative mortality in the EVAR group was 7.3% and the crude rupture rate in the no intervention group was 12.4 [95%CI9.6-16.2] per 100 patient-years, leading to lower aneurysm-related mortality in the EVAR group; adjusted hazard ratio 0.53 [95% CI 0.32-0.89], p=0.015. This advantage was not translated into any benefit in terms of all-cause mortality; adjusted hazard ratio 0.99 [95% CI 0.78-1.27], p=0.967. Incidence of complications after EVAR was high with approximately 50% of patients experiencing complications and 25% requiring re-interventions within the first 6 years. Thus, EVAR was considerably more expensive; cost difference £9,826 [95% CI £7,638-£12,103].
Conclusions: EVAR offered a significant long-term benefit over no intervention in terms of aneurysm-related mortality but not all-cause mortality. High rates of complications and re-interventions after EVAR contributed to increased costs for the EVAR group. The future treatment strategy for these unfit patients may be defined by cost-effectiveness evaluations.

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